What is claimed is:

1. A method of identifying an agent effective in modulating Stat3-dependent cell proliferation, said method comprising the steps of:
   i) incubating TEL/Etv6 with a compound;
   ii) detecting TEL/Etv6 activity; and
   iii) determining a compound-induced modulation in the TEL/Etv6 activity relative to when said compound is absent, wherein an alteration of the TEL/Etv6 activity in the presence of the compound is indicative of an agent effective in modulating Stat3- dependent cell proliferation.

2. The method according to claim 1, wherein said modulation is inhibition of TEL/Etv6 activity and said agent is effective in enhancing cytokine-induced inhibition of cell proliferation.

3. The method according to claim 1, wherein said modulation is activation of TEL/Etv6 activity and said agent is effective in inhibiting proliferation of cells expressing Stat 3, wherein said Stat3 is phosphorylated.

4. The method of claim 3, wherein said cell proliferation is independent of ras activity.

5. The method of any one of the preceding claims, wherein said cell proliferation is of a melanoma or carcinoma.

6. A method for identifying an agent effective in modulating Stat3-dependent cell proliferation, said method comprising the steps of:
   (i) incubating at least one TEL/Etv6 polypeptide selected from the group consisting of TEL/Etv6, a variant and a fragment thereof, with a binding partner in the presence of a test compound; and
   (ii) determining whether the presence of a test compound modulates the interaction between said TEL/Etv6 polypeptide and said binding partner relative to when said test compound is absent.

7. The method according to claim 6, wherein the variant or fragment of TEL/Etv6 has the ability to bind Stat3.

8. The method according to claim 6 or 7 wherein the fragment of TEL/Etv6 is between 50 and 350 amino acids in length.

9. The method according to any one of claims 6 to 8, wherein said binding partner is Stat3, a variant or fragment thereof.

10. The method of any one of the preceding claims, further comprising confirming that the test compound is a modulator of Stat3-dependent cell proliferation.

11. The method according to any one of claims 6 to 10, wherein said TEL/Etv6 polypeptide or the binding partner is labelled with a detectable label, and the other is immobilised on a solid support.

12. The method according to any one of the preceding claims wherein the modulation is inhibition of said interaction.

13. The method according to claim 12 comprising the step of confirming that the substance inhibits cell proliferation of a cytokine-sensitive cancer.

14. The method according to claim 12 or 13, comprising determining whether said test compound inhibits the physical association between TEL/Etv6 and Stat3.

15. The method according to any one of claims 6 to 14, said method comprising the steps of:

(i) contacting a cell expressing TEL/Etv6, a variant or fragment thereof which has the ability to interact with said binding partner, with a test compound, and

(ii) identifying substances which inhibit said interaction in said cell.

16. The method according to claim 15, said method comprising:

(i) providing a cell capable of expressing the TEL/Etv6 polypeptide and its binding partner and a reporter gene construct,

(ii) contacting the cell with a test compound,

whereby inhibition by the test compound of binding between the TEL/Etv6 polypeptide and the binding partner can be observed as a reduction of reporter gene expression.

17. A mammalian cell capable of expressing a TEL/Etv6 polypeptide, its binding partner, and a reporter gene construct, whereby binding between said TEL/Etv6 polypeptide and said binding partner can be observed by reporter gene expression.

18. A method of inhibiting Stat3 expressing cancer cell proliferation, said method comprising contacting a cancer cell expressing Stat3 with an effective amount of an activator of TEL in an amount sufficient to inhibit Stat3 activity.

19. The method of claim 18, wherein said Stat3 is phosphorylated.

20. A method of inhibiting cytokine sensitive cancers, said method comprising contacting a cytokine-sensitive cancer cell with an effective amount of an inhibitor of TEL activity in an amount sufficient to enhance Stat3 activity.

21. The method of claim 20, wherein the inhibition of activity is caused by down-regulating TEL/Etv6, or a homologue thereof in the cell.

22. The method of claim 21, wherein said down-regulation is caused by RNAi.

23. The method of claim 22, wherein said down-regulation is caused by an at least partially double-stranded RNA of between 20 and 25 bps in length, comprising an RNA sequence encoding a portion of TEL/Etv6 or a homologue thereof.

24. The method of claim 20, wherein said TEL inhibitor is an antibody or antibody fragment.

25. The method according to claim 20 or 24, wherein the inhibition of activity is caused by inhibiting the interaction of TEL/Etv6, or a homologue thereof with a binding partner in the cell.

26. The method according to claim 25 wherein the binding partner is Stat3.

27. Use of an at least partially double-stranded RNA comprising an RNA sequence encoding TEL/Etv6, a homologue or a fragment thereof, to inhibit cell proliferation of a cytokine-sensitive cancer cell.

28. Use of the double-stranded RNA according to in claim 27, wherein said dsRNA is an siRNA duplex of between 20 and 25 bps.

29. Use of an inhibitor of TEL/Etv6 activity in the preparation of a medicament for the treatment of a patient suffering from a cytokine-sensitive cancer.

30. Use of an activator of TEL/Etv6 activity in the preparation of a medicament for the treatment of a patient suffering from STAT3 expressing cancer.